

# Multiparametric Ultrasound Examinations in the Diagnosis of Intraventricular Cerebral Hemorrhages in Children

Gulnora Akmalovna Yusupalieva<sup>ID</sup>, Adiba Rustamovna Manashova<sup>ID</sup>, Laylo Rustamjonovna Sultanova<sup>ID</sup>, Umida Asqarovna Umarova<sup>ID</sup>, Shakhnoza Ortikboy Ortikboeva<sup>ID</sup>

Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan.

## Abstract

Intraventricular cerebral hemorrhages in children in the structure of pediatric morbidity in Uzbekistan account for about 8-15% of newborns (Statistical materials on the activities of healthcare institutions of the Republic of Uzbekistan in 2017). For the further development of perinatal neurology, it is necessary to search for new objective methods for assessing the functional and structural state of the brain. Topical diagnostics is possible with the help of radiation research methods. The present work was based on the results of a neurosonographic examination of 90 sick children with VVC. All patients underwent brain ultrasound using standard and polypositional neurosonography (stNSG and pNSG) techniques. All children in the hospital population had an increased risk of developing VVC. The ratio of newborns and children aged 1-12 months of life was approximately the same (52.5 and 47.5%), boys significantly prevailed (71.2%). More than half of the children (61.8%) were born from natural childbirth in the head presentation. In 15 children with grade II VVC, the CNS depression syndrome is more pronounced. There may be a disorder of consciousness, sopor, and in more severe cases – coma, 4 children have anomalies of pupillary reactions, 7 children oculomotor disorders were often combined with bulbar disorders (violation of sucking, swallowing, pathology of breathing and cardiac activity.) Differences with the comparison group were significant only in relation to the frequency of convulsive syndrome in 5 children.

**Keywords:** Intraventricular Hemorrhage, Newborn, Pediatric Morbidity, Osteodiasis, Shell Clusters.

**Corresponding Author:** Gulnora Akmalovna Yusupalieva, Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan.

E-mail: [Ygulnora@mail.ru](mailto:Ygulnora@mail.ru)

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## Introduction

Intraventricular hemorrhages (IVH) occupy a leading place in the structure of perinatal lesions of the nervous system in newborns and are one of the main causes of deaths, which account for 9-27% in full-term and up to 70% in premature infants. Hemorrhages suffered in 55.4% of cases cause neuropsychiatric disorders.<sup>[1-5]</sup>

Intraventricular cerebral hemorrhages in children in the structure of pediatric morbidity in Uzbekistan account for about 8-15% of newborns according to the Statistical materials on the activities of healthcare institutions of the Republic of Uzbekistan in 2017.

The main etiological factor of perinatal pathology of the nervous system and, in particular, VVC, is hypoxia, which leads to hemorrhagic lesions in newborns. Its result is a cerebrospinal fluid disorder, leading to brain dysfunction. This requires the dynamic observation of many specialists,<sup>[6,7]</sup> including ultrasound examination (ultrasound) of doctors to

determine the development of hydrocephalus in a timely manner.

For the further development of perinatal neurology, it is necessary to search for new objective methods for assessing the functional and structural state of the brain. Topical diagnostics is possible with the help of radiation research methods.<sup>[8,9]</sup>

However, the use of computed tomography (CT) and magnetic resonance imaging (MRI) is difficult, especially in the conditions of the intensive care unit and in preterm infants and for constant dynamic monitoring of the state of cerebrospinal fluid dynamics and hemodynamics.<sup>[10-12]</sup> All of the above is considered an urgent problem and requires further study.

### The purpose of study

Optimization of the diagnosis of intraventricular cerebral hemorrhages in children through the use of modern ultrasound research methods.

## Materials and Methods

The present work was based on the results of a neurosonographic examination of 90 sick children with VVC. All patients underwent brain ultrasound using standard and polypositional neurosonography (stNSG and pNSG) techniques. All children in the hospital population had an increased risk of developing VVC. The ratio of newborns and children aged 1-12 months of life was approximately the same (52.5 and 47.5%), boys significantly prevailed (71.2%). More than half of the children (61.8%) were born from natural childbirth in the head presentation. Almost a quarter of patients (22.2%) were born by caesarean section. Complicated situations in childbirth were often encountered: rapid labor took place in 4.0% of cases, secondary weakness of labor activity - in 4.0%, vacuum extraction - in 6.0% and labor in the buttock and leg presentation - in 3 (3.0%) cases. Birth trauma, as an etiological factor in the development of VVC, was diagnosed in 17.8% of cases, in the presence of clinical manifestations of this pathological condition (pain syndrome, general brain and focal neurological symptoms) and signs of mechanical damage to the skull (cephalomatoma, pathological configuration of the newborn's head, fractures of the skull bones, osteodystasia of the occipital bone).

Birth traumatic injuries were diagnosed in 12.9% of children without neurological symptoms with 1 art.

Hypoxic-ischemic lesion of the central nervous system was detected in 3% of children with IVF 3-4 ct, hypoxic-traumatic - in 4%.

Postnatal traumatic brain injury caused the development of VVC in 25.7% of children.

Household traumatic brain injury occurred in 20.8% of cases, as a rule, it occurred when children accidentally fell out of the hands of adults, from a changing table or bed. Traumatic brain injury in a traffic accident was registered in 1% of the patient.

In 1 child, multiple fractures of the skull were detected in combination with massive PVCs, a focus of brain contusion and severe damage to the parenchyma in the form of subcortical necrosis; in 3 other children, subacute bilateral IVCs were detected in combination with PVCs.

Comprehensive clinical, laboratory and instrumental examinations included a detailed history collection, physical examination, neurosonographic examination in the Republican Perinatal Center and on the basis of the Republican Scientific Center of Neurosurgery on Sonoscape 5000, Aplio 500 ultrasound diagnostic devices ("Toshiba" Japan), Mirror 2.

The control, i.e. comparison group consisted of 20 healthy children of the same age. In the control group, we examined practically healthy children.

High-frequency sector sensors 5.0 and/or 7.5 MHz were used to conduct ultrasound examination of the brain in newborns

and young children. When using convexic sensors, the view of the lateral structures of the brain is limited. The severity of the condition was not a contraindication for neurosonography. Scanning was carried out in B-mode, CDK mode and pulse Dopplerography.

All children underwent NSG in the first 1-2 days after admission to the hospital, additional preparation for the study was not required.

Polypositional NSG (pNSG) was performed in the position of the child lying on his back, followed by turning the head to the right and left. Patients in extremely serious condition who were on artificial ventilation were examined in the presence of a resuscitator.

The technique included polypositional scanning using both traditional access through the large fontanel, and accesses through the occipital, mastoid fontanelles, temporal access, sutures and through bone defects in fractures of the skull bones. Scanning was carried out in frontal, sagittal, axial and oblique planes using convexic and high-frequency linear sensors.

In addition to the study in the "gray scale" mode, Dopplerographic techniques were used. CDC was used to assess the vascularization of the shell spaces and the brain, and the mode of pulse Dopplerography made it possible to obtain quantitative indicators of cerebral blood flow and to identify disorders of cerebral hemodynamics.

The pNSG Protocol included the following items:

- Differentiation and symmetry of brain structures;
- The presence or absence of displacement of the median structures of the brain;
- Assessment of brain parenchyma (echogenicity, presence of diffuse and focal changes, pattern of furrows and convolutions);
- Dimensions of internal liquor spaces: lateral, III and IV ventricles;
- Assessment of vascular plexuses, ventricular ependyma, presence of inclusions in the cerebrospinal fluid;
- Dimensions of the external cerebrospinal spaces (width of the subarachnoid space along the convexital surface of the cerebral hemispheres, width of the interhemispheric gap);
- Presence or absence of pathological shell clusters (indicating localization, shape, size, echogenicity and structure);
- Patency of the liquor pathways;
- Assessment of the structures of the posterior cranial fossa: the symmetry of the cerebellar hemispheres, the size of the large occipital cistern, the presence of paracerebellar shell clusters, their size and structure, as well as the presence of echogenic inclusions in the lumen of transverse venous sinuses;

- Dopplerographic indicators of cerebral blood flow: maximum systolic blood flow velocity,  $V_{max}$ , in the anterior cerebral artery; minimum diastolic blood flow velocity,  $V_{min}$ , in the anterior cerebral artery; resistance index, RI, in the anterior cerebral artery; blood flow velocity in the Galena vein and transverse sinuses.

## Results and Discussion

When analyzing the features of the clinical course of VVC, we noted the relationship between the severity of neurological symptoms and the severity of hemorrhages. Neurological manifestations did not differ significantly in 5 children with grade I VVC and in 15 children in the comparison group. The time of occurrence of neurological symptoms varied from 1-2 to 8-9 days of life. In 5 children with grade I VVC, there was a moderately pronounced CNS depression syndrome, which is characterized by suppression of unconditioned reflexes, 4 children with a decrease in spontaneous motor activity, 6 children with a decrease in muscle tone and 2 children of tendon reflexes with oculomotor disorders. At 2-3 weeks of life, all children became more active, signs of hyperexcitability syndrome appear - increased spontaneous motor activity, revival of unconditioned reflexes, muscular dystonia and tendon hyperreflexia. Convulsive disorder was observed in 2 children of the I-degree of VVC in the neonatal period.

In 15 children with grade II VVC, the CNS depression syndrome is more pronounced. There may be a disorder of consciousness, sopor, and in more severe cases – coma, 4 children have anomalies of pupillary reactions, 7 children oculomotor disorders were often combined with bulbar disorders (violation of sucking, swallowing, pathology of breathing and cardiac activity.) Differences with the comparison group were significant only in relation to the frequency of convulsive syndrome in 5 children.

With grade III VVC, it was the most difficult, the children were in a comatose state for a long time, due to respiratory disorders, CVL was brought.

Bulbar disorders of 3 children were often noted, in 4 children the convulsive syndrome remained resistant to therapy for a long time. In 8 children, there was a sharp suppression of unconditioned reflexes, muscle hypotension, tendon hyporeflexia, bradycardia, decreased hematocrit, metabolic acidosis seizures. A combination of neurological symptoms was noted. A directly proportional dependence of the frequency and severity of structural changes in children in the first year of life on the degree of hemorrhage suffered in the neonatal period was revealed. In children after IVF of the III degree, by 9-12 months of life, a delay in the psycho-motor development of 3 children, cerebral palsy of 2 children, convulsive syndrome of 4 children were noted. The prognosis was more favorable in the group of children with grade I and II VVC.

During echography, grade I VVC was characterized as a hyperechoic rounded formation inside the ventricles or vascular plexus. In 5 children, the VVC was bilateral. A dynamic study revealed the formation of intraventricular pseudocysts after grade I VVC on day 10-14 in 84% of children. It should be noted that the thrombus in the lumen of the lateral ventricle in the initial stage of the disease was tightly attached to the vascular plexus and, possibly, to the germinal matrix, although not differing from them in structure. Thus, it is very difficult to accurately measure the size of a blood clot, usually it was captured together with the vascular plexus and the germinal matrix.

The thrombus located in the cerebrospinal fluid pathways has undergone significant changes over time. In the first 5-7 days, it had a fairly homogeneous echogenic structure, and then lysis processes occurred. The thrombus became heterogeneous, the echogenicity of its middle part decreased, as a result of which the contours looked underlined. Further, the thrombus fragments or gradually resolves with a change in shape, size, consistency.

With grade II VVC, nodular thickening of the vascular plexus, its asymmetry and expansion were noted, the expansion of the lateral ventricles on the hemorrhage side was determined, the depth of the lateral ventricles at the level of the bodies was more than 5 mm. within normal limits. In 2 children, the VVC was bilateral. During the course of the II degree of VVC, the state of the cerebrospinal tract changed, posthemorrhagic ventriculitis often developed, which during ultrasound was represented by a combination of three main echosymptoms: - ventriculomegaly; - increased echogenicity of the walls of the lateral ventricles; - the presence of a fine suspension in the lumen of the lateral ventricles. In uncomplicated cases, ventriculomegaly increased for 2-3 weeks, after which, against the background of treatment, the condition stabilized, and after 1 month, positive dynamics was noted.

Grade III VVC was characterized by dilation and nodular thickening of the vascular plexus, which was accompanied by ventricular dilation and parenchymal hemorrhage. During 2-3 weeks, the echogenicity of the parenchymal hemorrhage area gradually decreased, only local hyperechoic inclusions remain. After 3-4 weeks, at the site of parenchymal hemorrhage, there were formations of a porencephalic pseudocyst in the form of an anechoic structure with clear contours communicating with the lateral ventricle of the brain. In 8 children, clots formed that repeated the shape of the ventricles of the brain, causing their tamponade. There was an increase in ventriculomegaly from minor to severe with the formation of internal hydrocephalus. In 2 children, the VVC was bilateral.

In many cases, high-grade VVC was accompanied by hemorrhage into the cavity of the III- IV ventricles. The III ventricle was optimally visualized when scanned through the scales of the temporal bone, while in the acute stage of the process, the

blood in it looked like the contents of increased echogenicity. Gradually, the clot was transformed in the same way as blood clots in the lateral ventricles: its middle part turned out to be hypoanechogenic, and the contour was emphatically echodense.

A formidable complication of high-grade VVC was hemorrhage in the posterior cranial fossa. It is quite difficult to visualize it with NSG, it is advisable to use non-standard scanning accesses. The most frequent finding was a hemorrhage in a large cistern. Fresh blood looked like the contents of increased echogenicity, evenly filling the entire large tank. Its size in the acute phase of the disease usually did not increase. Quite often, a block of the cerebrospinal fluid pathways developed with high-grade VVC, while in the acute phase of the disease, a sharp increase in dilatation of fragments of the cerebrospinal fluid pathways was determined. In their lumen, it was sometimes possible to identify fragments of blood clots. The block of the cerebrospinal fluid pathways sometimes arose due to the adhesive process or against the background of the addition of inflammatory changes. The narrowest places were most often affected: the Sylvian water supply, the Monroe hole. With the block at the level of the Monroe orifice, a significant dilation of the lateral ventricle on the side of the block developed. With the block of the Sylvian aqueduct, the sizes of both lateral and III ventricles sharply increased.

There was a block at the level of a large tank, which was accompanied by a significant, sharp deterioration in the condition of the child with a violation of vital functions. The cerebrospinal fluid pathways were dilated throughout, and the large cistern acquired a spherical shape, sharply pushing the medulla oblongata in the ventral direction. Grade III with neurosonography was diagnosed quite easily: the parenchymal component was pathognomonic for this type of hemorrhage, i.e. it is determined not only a thrombus in the lumen of the lateral ventricle, but also the focus of hemorrhage in the parenchyma of the brain, adjacent directly to the thrombus in the lumen of the ventricle. Ventriculomegaly developed dynamically, and hemorrhage in the parenchyma of the brain led to the formation of a large porencephalic cyst. The parenchymal component of grade III VVC occurred in any part of the brain adjacent to the lateral ventricle, while in all cases the contour of the lateral ventricle was not traced. In massive hemorrhages, the dislocation of the median structures of the brain shifted in the direction opposite to the hemorrhage with a sharp violation of the anatomy of the brain.

In addition to the In-mode study, a Doppler evaluation of cerebral hemodynamics was performed in all patients with ultrasound signs of IVC.

The CDC mode with the assessment of visualization of the vascular pattern of the brain and the pulse Dopplerography mode with the possibility of quantitative assessment of cerebral hemodynamics were used. The parameters of blood

flow in the anterior cerebral arteries, Galena vein and transverse sinuses were studied.

#### **The following symptoms were identified:**

A symptom of vascular pattern flickering. This sign was recorded in the CDK mode with a marked decrease in the rate of arterial blood flow in the diastole and a significant increase in vascular resistance. The change in the color of the arterial blood flow staining from red to blue indicates the appearance of a reverse flow in the diastole, which corresponds to the values of the resistance index of more than 1.0. The symptom of vascular pattern flickering was detected in 9.8% of children with ultrasound signs of VVC. In all cases, the condition of the children was regarded as very serious, all of them were being treated in the intensive care unit.

The symptom of an increase in the resistive characteristics of blood flow in the cerebral arteries is, in fact, the quantitative equivalent of the flicker symptom. Taking into account the urgency of the situation and the generalized response of the infant's cerebral hemodynamics to VVC, the measurement of quantitative characteristics of blood flow was carried out in the anterior cerebral artery (PMA), less often in the pericallosal artery. The symptom of an increase in the resistive characteristics of blood flow was recorded at values of the resistance index (RI) in the PMA of more than 0.7. The symptom was detected in 39.0% of children with ultrasound signs of VVC. In the acute period of massive VVC and PVK, there was a sharp increase in the resistance index in PMA to 1.0 and higher, which was observed in 9.8% of children. In 3.6% of cases, there were modulations of the amplitude of arterial blood flow depending on the act of breathing, which was regarded as a sign of impaired autoregulation of cerebral blood flow. A decrease in the resistance index in the acute period was not detected in any case.

## **Conclusion**

Thus, pNSG allowed to diagnose VVC not only in B-mode, but also using Doppler blood flow assessment techniques.

Summarizing the study of the echographic semiotics of VVC in newborns and children of the first 3 months of life, it can be concluded that stNSG, with which the examination of each infant begins, does not always allow to identify the main echosymptoms of VVC and reliably diagnose pathology. With stNSH, it is possible to visualize only large VJCS and identify their indirect signs, while PNSH allows you to visualize not only large VJCS not detected with stNSH, but also small VJCS.

A change in the parameters of cerebral blood flow cannot be considered a sign of VVC, but reflects a non-specific reaction of cerebral hemodynamics to an increase in intracranial pressure during VVC and characterizes the overall severity of



brain damage. The Doppler study of cerebral blood flow plays an auxiliary role in the diagnosis of VVC, but undoubtedly has an important prognostic value.

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